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Abstracts

Gene regulation

Program/Abstract # 139
Quantitative dissection of a repressive morphogen gradient
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The gap gene *hunchback (hb)* is critical for patterning the anterior–posterior (AP) axis of the early *Drosophila* embryo. *hunchback* encodes a Zn-finger transcription factor, and forms a protein gradient in the middle region that refines dramatically over time. Previous work suggests that Hb activates and represses different target genes. Its repression targets include *Kruppe (Kr)*, *knirps (kni)*, *giant (gt)*, *nubbin (nub)*, *POU domain protein 2 (pdm2)*, and the enhancers that drive expression of *eve* stripes 3 and 4. By simultaneously imaging the RNA expression patterns of pair-wise combinations of target expression patterns, we precisely defined their relative positions along the AP axis. We then simultaneously image each target RNA expression pattern with the Hb protein gradient, and analyze this relationship over time. These experiments suggest that most target genes shift in concert with the refining gradient, which permits the calculation of the relative Hb concentrations that correspond to the position of each repression event. We then use a ventral expression system to test whether Hb concentration alone can account for the differential positions of the different target genes. Our results suggest that those target genes that shift over time with Hb are differentially sensitive to specific thresholds of Hb concentration. They further define limits for the range of protein concentrations that mediate morphogenetic activity. We show further that two target genes positioned outside this morphogenetic range each use combinatorial repression mechanisms involving Hb and other gap proteins.

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Program/Abstract # 140
The possible interaction between ORF2 and TFIIH in Drosophila melanogaster
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TFIIH, a basal transcription factor, also participates in DNA repair and cell cycle control. TFIIH is composed of 10 subunits divided in two subcomplexes: the Core (XPD, XPB, p62, p52, p44, p34 and p8) and the CAK subcomplex (Cdk7, CycH and Mat1). Mutations in some TFIIH components can lead to three hereditary human disorders known as Xeroderma Pigmentosum, Cockayne Syndrome and Trichothiodystrophy (TTD). The genetic cause for defective DNA repair in a photosensitive form of TTD (TTD-A) is a mutation in p8. Since p8 overexpression restores the reduced cellular level of TFIIH in TTD-A patients cells, it has been proposed that p8 contributes to the stability and concentration of TFIIH. We searched for the *Drosophila* homologue of p8 and found that it is encoded in a bicistronic transcript. The second open reading frame encodes a protein of 150 amino acids and we named this putative protein ORF2. Interestingly, ORF2 has a HIT zinc finger domain and is conserved in all eukaryotes. Moreover, there are examples of functional relation between proteins encoded by bicistronic transcripts. Therefore, it is possible that ORF2 could be part of TFIIH or can interact with a component of it. To determine the possible interaction of ORF2 with TFIIH we have generated transgenic flies that express tagged ORF2 or p8. These flies will be used to perform co-immunoprecipitations using antibodies against the tags, as well as other TFIIH components. With these tools we plan to determine the subcellular localization of these proteins during fly development. These strategies will help us to propose a role for ORF2 with TFIIH.

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Program/Abstract # 141
Developmental defects caused by mutations in the p52 subunit of TFIIH in Drosophila mimic human diseases
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